MEDICAL
PRACTICE OF
PREVENTIVE
NUTRITION

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MEDICAL PRACTICE OF PREVENTIVE NUTRITION

Edited by Mark L. Wahlqvist and Jitka S. Vobecky

Throughoutout medical history, there has generally been appreciation that there is value in the use of food, or more recently nutrients, in therapeutics, and there has been acceptance that, since food is vital to life, its wise use prevents health problems. A more systematic examination of how particular nutrients, foods or food patterns might contribute to major disease problems, and therefore how changes in usage might allow prevention, began with the identification of nutrient deficiency disorders, but in the last 20 - 30 years, diet has become the focus of attention in relation to the major non-communicable diseases of people in industrialized and industrially emerging nations. The suggested opportunities for improved quality of life where there is less illness and greater sense of well-being, and which may be possible with changes in nutritional status, are themselves a stimulus for preventive nutrition.

Preventability of the disease need not be absolute or complete avoidance of the disease. It may be the delay in onset of disease until a later age. The severity of the disease may also be reduced. Prevention also has to do with the maintenance or enhancement of well-being. It is important to identify and take opportunities for prevention in primary (where no disease exists), seondary (where the basic disease process has manifested itself) and tertiary (where complications of the disease in question have arisen) settings. Reversability of disease processes is also another dimension of prevention.

How does the approach of using nutritional means to prevent disease fit into medical practice? What are the limitations of this approach and what needs to be known before embarking on it seriously? After assessing these questions, the bulk of the book considers the application of the preventive approach to nutrition in a range of systemic diseases. A critical approach is designed to elicit greater awareness of the various facets of prevention and avoids prescribing facile 'answers'. The book will serve as a practical reference manual to guide practitioners and support teachers of the subjects involved in developing their own, appropriate measures of successful preventive nutrition practice.

WAHLQVIST
VOBECKY

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This manual is conceived and developed by the IUNS Committee on
Medical Education under the Chairmanship of Professor Jitka Vobecky

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Introduction

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Acceptance of prevention through nutrition

Throughout medical history, there has generally been an appreciation that there is value in the use of food, or more recently nutrients, in therapeutics, and there has been an acceptance that, since food is vital to life, its wise use prevents health problems. A more systematic examination of how particular nutrients, foods, or food patterns might contribute to major disease problems, and therefore how changes in usage might allow prevention, began with the identification of nutrient deficiency disorders, but, in the last 20–30 years diet has become the focus of attention in relation to the major non-communicable diseases of people in industrialized and industrially-emerging nations. Indeed, one of the greatest challenges may now be the approach that should be taken where nutritional status is transitional between protein and specific nutrient deficiency on the one hand and energy imbalance with consequent overfatness on the other.

The suggested opportunities for improved quality of life where there is less illness and greater sense of well-being and which may be possible with changes in nutritional status are themselves a stimulus for preventive nutrition. Once day-to-day survival ceases to be a problem for the poor and hungry, the quality of food supply and healthy food choices can be contemplated. Even so, one is often quite culturally-bound in the food one chooses – and there are historical examples of how people have died where food was available, but not acceptable. The Caucasian Australian explorers, Burke and Wills, perished, in an arid area, with no residual food supplies, but where Aboriginal Australians lived on local bush food. Interestingly, recent work by Ono in Japan indicates that there is specific neuronal memory for food[1,2]. The ability to distinguish between food and non-food is found in the amygdalal part of the brain, so that neurophysiological factors (amongst others) may need to be taken into account, or taken advantage of, in preventive medicine. Furthermore, equity in food distribution, competent food production, storage and processing, sufficient food knowledge and safe food behaviours generally need to be in place before quality of life and long-term health can become personal food choice issues.

In difficult economic times, governments and individuals are driven increasingly

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by considerations of cost-effectiveness. Health departments and agencies see that if chronic, disabling diseases like stroke, diabetes and osteoporosis with fractures can be prevented by nutritional means, then costs to the community will be saved and life-long productivity increased.

Nature of preventability

Preventability of disease need not be absolute or complete avoidance of disease. It may be the delay in onset of disease until a later age. If, for example, the menopause is partly nutritionally determined[3], and it is delayed or modified, then significant osteoporosis and related fracture may be delayed. Again, if biological aging occurs at an older chronological age, as appears to have happened in the last few decades in Sweden and presumably elsewhere, (See chapter 9 and[4]) then this could be an important way in which eating patterns might contribute to prevention.

The severity of disease may also be reduced. For example, if coronary stenosis due to atherosclerosis is lessened, the chances of thrombotic occlusion reduced, or the likelihood of arrhythmia with reduced myocardial blood flow decreased, and each of these is separately influenced by nutritional factors, the severity of ischaemic heart disease is less and, in measure, preventable[5]. Prevention also has to do with the maintenance or enhancement of well-being.

It is important to identify and take opportunities for prevention in *primary* (where no disease exists), *secondary* (where the basic disease process has manifested itself) and *tertiary* (where complications of the disease in question have arisen) settings. *Reversibility* of disease processes is also another dimension of prevention.

Assumptions in prevention strategies

A judgement is required about present evidence for prevention and the risks and benefits of action. The assumptions underlying preventive nutrition include the following:
1. That the disease process is modifiable.
2. That, in a multifactorially determined disease process, changing a particular factor can be critical or, if not, that several factors may require change.
3. That the theoretical basis for prevention is often incomplete and, usually, as with other biomedical approaches, based on composite lines of evidence, with, on occasion, extrapolation to the situation of interest.
4. That the way we eat has a significant role in the disease process in question.
5. That there are certain preferred ways of eating, and limits to the way we can eat, if optimal health and maximal life span are sought.
6. That what we do at one age may be biologically consequential for a later age, or that it may be worth doing in later life.
7. That what is necessary to change is capable of definition, and that the process of change is sufficiently straightforward and realistic to stand a chance of being carried through, taking account of cultural and other constraints.

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BODY HABITUS

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Introduction

Body composition changes can be both markers of biological development and indicators of disease processes which are common, such as obesity, non-insulin-dependent diabetes mellitus, vascular disease which are associated with over-nutrition, and marasmus/kwashiorkor, malignancy, and chronic disease which are associated with undernutrition. Methods of measuring and following these changes have been developed with varying levels of portability, simplicity, precision and cost. These techniques are available both in hospitals and for epidemiological surveys. They provide a method at an individual and community level of assessing nutritional status and response to treatment, and have a potential for prevention of these conditions.

What is meant by body habitus or body composition?

Most people have a concept of a range of normality with respect to body weight. This involves a recognition of obesity and wasting as abnormal states associated with health impairment. Older concepts of this 'body habitus' included the terms 'ectomorph', to define thinner individuals, 'endomorph' to define broader individuals, and 'mesomorph' to define those whose build fell in between these extremes. Today, these concepts have been redeveloped to describe fat mass, fat distribution, lean mass and skeletal mass as part of a newer physiological concept of 'body composition' being a determinant of health and disease. An understanding of body composition changes has come about because of new technologies enabling these compartments to be measured.

The measurement of body composition

The measurement of components of body composition (BC), correlating these structural changes with function, and following the changes over time as a result of normal changes, disease, training or therapy has become more widely discussed over recent years.[1]

The two compartment model of body composition

The two compartment model of body composition defines a fat compartment and a lean compartment, and is the oldest of the models[2]. Essentially, it is based on the

separation of water-miscible and lipid containing components of the body. This is a simplistic model in that all cell membranes contain lipid, and such a separation is probably artificial. There is some confusion about the terms which are used within this model.

Fat mass (FM) is defined as the mass of stored triglyceride, phospholipid and circulating fatty acids in the body, and is thus truly water-free.

The fat-free mass (FFM) is the remainder of the body weight after subtraction of the fat mass.

Both of these compartments are physiological concepts. The adipose tissue (AT) compartment consists of the mass of stored triglyceride associated with adipocytes, its supporting connective and vascular tissue, and the surrounding extracellular water.

The lean body mass (LBM) consists of the nonadipose tissue compartment of the body, including the skeleton and extracellular connective tissue, and includes the lipid component of the cell membranes included in this mass.

The terms lean body mass and fat-free mass, and adipose tissue and fat mass are often used interchangeably, but it can be readily seen that there is a small but significant degree of overlap.

The four compartment model of body composition

The four compartment model involves measuring three subdivisions of the FFM, in addition to assessing the FM[3].

Total body water (TBW) is the volume of water in which the LBM exists. It has both an intracellular (ICW) and an extracellular (ECW) component.

Total body protein (TBP) is the mass of protein found both extra- and intracellularly.

Total body calcium (TBC) effectively measures the mineral component of the skeleton.

How can the components of body composition be measured. What level of sophistication is required?

All clinical methods of body composition are approximate, since the absolute assessment of chemical analysis is unavailable, and, in any case, has been attempted on very few occasions[4].

The methods of body composition analysis which are of clinical use vary in their availability, portability, precision simplicity and cost.

ANTHROPOMETRY

This is the most widely used form of assessment, and is used directly to measure growth and fat distribution, and, by comparison with other methods, to derive FM and FFM. It is a method well suited to epidemiological studies as well as bedside assessment in the very sick individual.

The components able to be measured include height, weight, circumferences and skinfold thicknesses. Some of these parameters can be measured very precisely, to less than 1%, whilst other components are subject to more intra- and inter-observer variation[5].

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IMPEDANCE

The impedance of the body to a weak alternating current (800 µA, 50 Hz) is proportional to TBW. Recent development of bioelectrical impedance (BEI) has confirmed its value in the study of selected healthy populations and individuals, with a small error; when TBW shifts occur as a result of disease or therapy, the results are less accurate and less reproducible[6].

Most of the other techniques which are available tend to be located within hospital or laboratory research areas, and are hence more suited to use in individuals. Whilst the equipment is generally expensive to purchase, maintain and operate, more of the components of BC can be assessed, with very small disposable costs per test, at an acceptable level of invasibility.

UNDER-WATER DENSITOMETRY

This has been a 'gold standard' measurement of the two compartment model for many years. It is not a very practical instrument for measuring BC in sick or elderly individuals; there is an underlying assumption that the density of the FFM or LBM is uniform, and comparable between individuals, which is not true[7].

DEUTERIUM OXIDE DILUTION

This is beginning to replace tritiated water dilution as a relatively noninvasive 'gold standard' for measuring TBW; salivary assessment will make it acceptable to small children[8]. The technique may be useful in field studies where sample collection may take place, with later laboratory analysis.

TOTAL BODY POTASSIUM MEASUREMENTS

Such measurements using a whole body gamma counter are also acceptable for measuring change in children, and, together with TBW measurements by deuterium oxide dilution may provide an approximate measurement of ECW if it is assumed that all potassium is located intra-cellularly[2].

DEXA

Dual energy x-ray absorptiometry is a recent addition to the BC analysis field. Originally developed to quantify bone mass and bone density, this rapid technique, which involves a very low (0.02 mSv) x-ray exposure, is able to quantify regional and whole body AT, LBM and TBC. It has a very small error for measuring bone mass, whilst the error in measuring regional, especially abdominal, and whole body AT and LBM is beginning to be documented[9].

NEUTRON ACTIVATION ANALYSIS

This tool is potentially able to provide a simultaneous measurement of the whole body content of a range of elements; in practice, only nitrogen measurements, to assess TBP, have been developed, because of the radiation exposure required[10].

A number of other imaging tools enable body composition measurements; they include CT scanning[11], with the possibility of further defining fat distribution, and magnetic resonance imaging[12], with a potential for measuring intracellular elemental content; in practice these modalities are often fully utilized in performing scans for other disease processes.

Changes in body composition with age

An understanding of the risk or the actual changes of body composition in disease requires knowledge of normal variation.

Throughout life, body composition is always in a dynamic state. Major changes take place from infancy to childhood and adolescence. In normal individuals, changes in body habitus from late adolescence to old age continues at a less rapid rate. The body habitus profile in later life is different from that of the young adult[13,14].

The various components of body composition, including height, weight, LBM and AT are under genetic influences[15]. Pregnancy is accompanied by body composition changes in the mother, with an increase in ECF and ICF and AT and AT distribution, whilst the composition of the fetus varies throughout development[16]. Increases in body weight during pregnancy have been generally encouraged, although there are recent suggestions that the extra energy requirements of pregnancy have been overestimated. During pregnancy, 40% of total gain is represented by the fetus, placenta and amniotic fluid. There is a relationship between maternal weight gain and the weight of the new born infant. Inadequate early weight gain, defined as < 2.4kg by the 24th week of gestation, is associated with a significantly increased risk of intrauterine growth retardation. This risk is not diminished even when later weight gains have increased the total maternal weight gain to within recommended target levels[17].

The onset of puberty is accompanied by a spurt increase in LBM, especially in boys, and an increase in AT, especially in girls[18]. During adult years, AT varies much more than LBM and accounts for much of the variability in body weight. In late adulthood, there is a decline in LBM[19].

The factors most significant in predicting adult body habitus are childhood and parental body habitus. The Danish Adoption Study[20] showed that the risk of obesity is significantly increased if there is biological parental obesity, and that the relative contribution of environment was small. Some studies show that over weight infants tend to become over weight children, adolescents and adults. Height is an important determinant of adult LBM and TBC, as is ethnicity, in a manner independent of height. TBC is higher in American Blacks than in Caucasians, whilst Asians have a smaller LBM than Caucasians[21].

In the elderly, numerous mechanisms influence changes in body composition through the ill-understood aging process. Reduced efficiency of the DNA repair mechanisms, flaws in the synthesis of protein, impaired enzyme functioning, deterioration of immunological processes, actions of free radicals as well as possible cross linkage of proteins may contribute these changes[22,23].

Food and nutrient intake may be reduced through problems of dentition, an increased prevalence of gastro-intestinal disease, the effects of stroke and arthritis in the elderly. This may be potentiated by a reduction of appetite due to altered taste and smell sensitivity.

The usual changes in body composition associated with this aging process is a progressive decrease in fat free mass (FFM) and an increase in fat mass (FM). Epidemiological studies show an average % decrease in FFM per decade, averaging a total loss of 5 kg for women and 12 kg for men between the ages of 25–70 years[24]. Concomitantly, a 2 % increase in fat mass (FM) per decade after the age of 30, usually leads to a total increase of 10–15% of total body fat during an adult's life

span[25]. In the elderly, there is a shift of the TBW pool with a relative increase in the ECW, even in the apparent absence of cardiac, renal or hepatic disease, all of which may increase the ECW. This may be the result of a cell membrane process[26]. Body fat tends to slowly increase between 25–45 years, and continues to accumulate until 70–75 years[27], as shown in Figure 1.
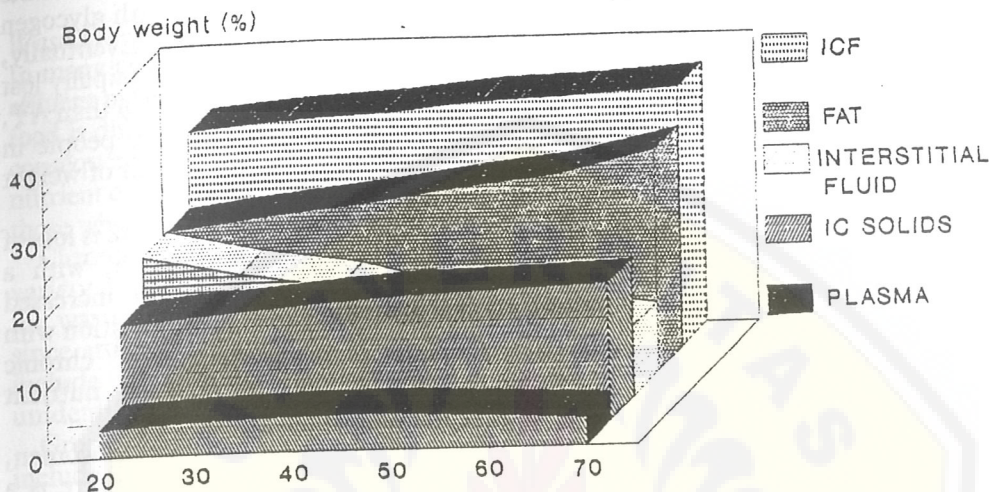

Figure 1. Changes in body composition with increasing age.

Health significance of changes in body habitus

The clinical significance of assessing body composition is the association between changes in composition with various diseases. Changes in body composition and fat patterns may be associated with the occurrence of various physiological functions that affect metabolism, nutrient intake, physical activity, and risk for chronic disease[28-30].

The health risks associated with the increased AT and LBM of obesity have been appreciated for many years. These include an increase in all-cause mortality, cardiovascular disease processes such as hypertension and coronary artery disease, endocrine disturbances such as non-insulin-dependent diabetes mellitus, hirsutism and infertility, gallstone formation, osteo-arthritis, breast and endometrial malignancy, and a variety of psycho-social disturbances[31].

In longitudinal and cross-sectional studies, there is relationship between an increase in percentage body fat, to atherosclerotic disease such as hypertension, hypercholesterolemia, hypertriglyceridemia. Conversely, a reduction in body weight has been shown to reduce blood pressure, especially in patients who suffer from mild hypertension. This nonpharmacologic approach has been widely employed for the management of patients with essential hypertension[32-34].

Recently, it has been shown that many of these risks are increased further when fat is distributed abdominally rather than around the buttocks, although it is still unclear whether this is a function of increased mesenteric fat, abdominal subcutaneous fat or both. Differing metabolic profiles of adipocytes from these sites have

been demonstrated[35]. Possible pathophysiological mechanisms include the release of free fatty acids FFA from intra abdominal fat directly into the portal vein. The high portal FFA concentrations then inhibits uptake of insulin by the liver, and leads to an increase in blood pressure, as well as the development of diabetes mellitus via peripheral hyperinsulinaemia.

The response to reducing energy intake to a level below that of expenditure results initially, in a release and excretion of the ICW water which is bound with glycogen stores, which are not assessed in any of the body composition models. Eventually, both AT and LBM are reduced, although the site from which AT is principally lost cannot be predicted; on occasions it is possible to lose relatively more LBM than AT, giving rise to a form of protein/energy malnutrition. In general, thin people in negative energy balance lose a relatively greater amount of LBM per unit of weight loss than do obese people.

In protein-energy malnutrition, with or without a reduction in AT, there is loss of TBP and distortions in the TBW, resulting in immunosuppression, with a significantly increased risk of infection, poor wound healing, and increased mortality. This condition is commonly found in major hospitals, in association with anorexia[36], chronic inflammatory bowel disease, short bowel syndrome,[37] chronic obstructive pulmonary disease[38], and alcohol abuse[39]. Usually, other nutrient deficiencies are also seen.

The skeletal mass continues to grow into the third decade of life, and, in women, undergoes some perimenopausal involution[40]. Osteoporosis, in which there is a reduction of TBC, carries a major risk for bone fracture in postmenopausal women and very elderly men[40].

Apart from defining these clinical processes, body composition measurements enable progress to be followed over time. The components of weight gain or loss in patients with burns, on TPN or following gastric reduction surgery are of clinical importance.

Such information is very essential for establishing health planning in a population, or establishing appropriate observations, and treatment evaluation in individuals and groups.

Who is at risk of body composition changes associated with disease?

Obesity

Whilst the basic cause of obesity is the result of a positive energy balance other nonmetabolic factors include both genetic and environmental factors. Parental weight adds to the risk of obesity[41]. Genetic predisposition for the development of obesity is higher in adult males[42]. A male with a childhood history of obesity has an 86% chance for developing obesity, whilst the figure for females is 80%.

Goldblatt et al.[43] studied the relationship between obesity and socio-economic factors amongst 1600 subjects in Manhattan who took part in an epidemiological survey of mental illness. There was an inverse correlation between obesity and socio-economic status. Amongst low socio-economic status women, the prevalence of obesity was six times more common. Amongst those women in the middle socio-economic group, the prevalence of obesity was three times higher. In these groups, high energy density foods are easily available, and may be seen as desirable.

Non-insulin-dependent diabetes mellitus (NIDDM) affects about 3% of the world's population, with particular ethnic groups, such as the Polynesians, being more susceptible. There is a strong genetic predisposition, but this predisposition is exacerbated by the presence of abdominal and general obesity. Improved glucose tolerance is associated with reduction in fat mass[44].

Wasting

In many parts of the world, the poor are systematically leaner than the rich and more affluent members of society. This difference is possibly due to energy restriction where food is difficult to grow, obtain or afford, and food knowledge is limited. The clinical consequences are growth retardation, marasmus, marasmic kwashiorkor, and micro-nutrient deficiencies, resulting in high infant mortality, persistent growth retardation in those who survive, and a high incidence of a variety of infectious and infestations.

Chronic gastro-intestinal disorders, such as inflammatory bowel disease, and a variety of malabsorptive conditions can lead to protein/energy malnutrition, whilst the wasting which is characteristic of advancing malignancy has been documented since at least Hippocratic times. The mechanisms involved in malignant wasting include altered food and taste perception, nausea, and vomiting, as well as unidentified metabolic factors.

Groups in whom food intake can be impaired to the extent that wasting occurs include those with anorexia and depressive psychological states, and those unable to feed themselves, which may occur in stroke, other chronic neurological conditions and severe arthritis (Figure 2).

——— Who is at risk ? ———

NIDDM	Anorexia/Bulimia
Childhood obesity	Physical disability
Parental obesity	Inadequate food intake
Over weight	Chronic GIT disease
Low socio-economic status (western society)	Poor dentition
	Malignancy
	Poor food knowledge
	Low socio-economic status (3rd world)

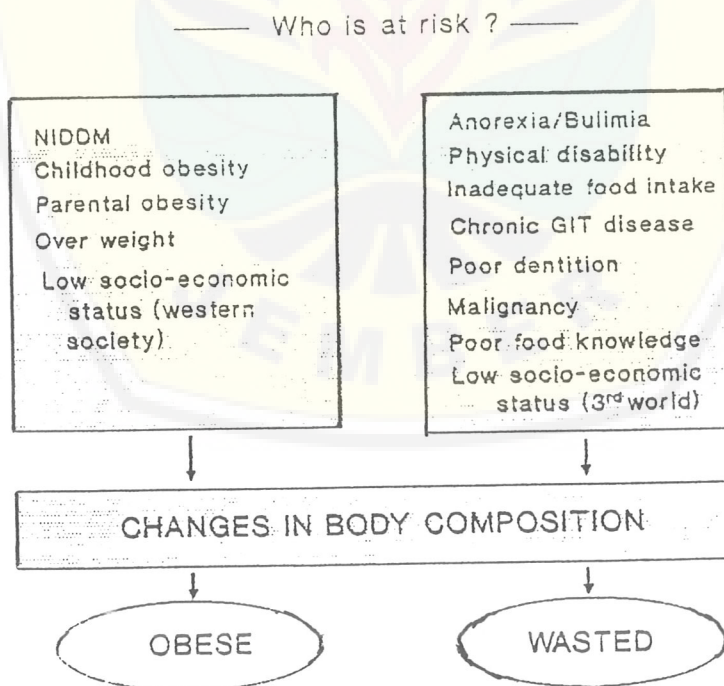CHANGES IN BODY COMPOSITION

OBESE   WASTED

Figure 2. Who is at risk of body composition changes associated with disease?

Osteoporosis

Osteoporosis occurs more commonly in women in whom menopausal hormone withdrawal interacts with genetic predisposition and nutritional status. Skeletal development depends on physical activity, whether cigarette smoking occurs, and on food habits, and continues into the third decade of life, which is well past the age at which most individuals consider that their growth has ceased.

Prevention of obesity, wasting and osteoporosis

In the individual

An important role for health workers is the identification of those who are at risk for developing obesity and its consequences, prior to the fat mass increase. This includes early counselling of those who have a family history or ethnic risk of obesity, and NIDDM, and vascular disease about their risk, and suggesting food habits and lifestyle habits which are likely to minimize their genetic predisposition. The early counselling should involve the family, including children at risk.

The food habits which need to be understood include knowledge about relative nutrient and energy density of foods, within the appropriate cultural confines, whilst physical fitness through daily activities needs to be encouraged.

An important component of the management of chronic illness likely to lead to wasting is nutritional status in its own right, so that the underlying process is treated without being exacerbated by wasting. Where the problem of wasting is associated with reduced availability of food, education about farming methods, and food habits becomes both an individual and community priority.

With the availability of new technology for assessment of body composition (see above), screening for asymptomatic osteoporosis in selected individuals at risk is possible. This assessment has the potential to detect reduced bone mass allowing early intervention to reduce fracture incidence in post menopausal women.

In the community

Public health recognition of the health risks associated with differences in socio-economic state and in different ethnic groups forms the basis of public education. This begins at school level, where facts and habits can be learnt to reinforce family lore.

Periodic epidemiological evaluation of body composition in community groups forms part of the public health knowledge base. This has been carried out for many years in children, but needs to be followed by adolescent and adult studies.

There is not yet an indication that bone density screening in the community will reduce the incidence of fracture in high risk women[45] although an intervention program which managed the prevention of excessive bone loss in a high risk group of postmenopausal women by administering hormone therapy has been shown to be very effective[46]. The benefit of a program should take into account the potential cost and benefits.

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